Welcome to DialogClassic Web(tm)

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 Dialog level 05.17.01D
Last logoff:  18jun07 12:34:23
Logon file1 21jun07 14:48:43
        *** ANNOUNCEMENTS ***
                  ***
NEW FILES RELEASED
***BIOSIS Previews Archive (File 552)
***BIOSIS Previews 1969-2007 (File 525)
***Engineering Index Backfile (File 988)
***Trademarkscan - South Korea (File 655)

RESUMED UPDATING
***File 141, Reader's Guide Abstracts
                  ***
RELOADS COMPLETED
***Files 154 & 155, MEDLINE
***File 5, BIOSIS Previews - archival data added
***Files 340, 341 & 942, CLAIMS/U.S. Patents - 2006 reload now online
                              ***
DATABASES REMOVED

Chemical Structure Searching now available in Prous Science Drug
Data Report (F452), Prous Science Drugs of the Future (F453),
IMS R&D Focus (F445/955), Pharmaprojects (F128/928), Beilstein
Facts (F390), Derwent Chemistry Resource (F355) and Index Chemicus (File 302).
                  ***
 >>>For the latest news about Dialog products, services, content<<<
 >>>and events, please visit What's New from Dialog at <<<
 >>>http://www.dialog.com/whatsnew/.  You can find news about<<<
 >>>a specific database by entering HELP NEWS <file number>.<<<
>>>PROFILE is in a suspended state.
>>>Contact Dialog Customer Services to re-activate it.
 * * *

File   1:ERIC 1965-2007/May
       (c) format only 2007 Dialog

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?

B 155, 5, 73
       21jun07 14:49:00 User259876 Session D1016.1
            $0.96    0.276 DialUnits File1
     $0.96  Estimated cost File1
     $0.06  INTERNET
     $1.02  Estimated cost this search
     $1.02  Estimated total session cost   0.276 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1950-2007/Jun 19
        (c) format only 2007 Dialog
 *File 155: Medline has been reloaded. Please see HELP NEWS 154
for information on 2007 changes.
  File   5:Biosis Previews(R) 1926-2007/Jun W3
        (c) 2007 The Thomson Corporation
 *File   5: BIOSIS has been enhanced with archival data. Please see
HELP NEWS 5 for information.
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  File  73:EMBASE 1974-2007/Jun 14
        (c) 2007 Elsevier B.V.

      Set  Items  Description
      ---  -----  -----------
?

S (TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
         195085  TRANSGENIC
         185235  DROSOPHILA
          52105  ELEGANS
         180222  ALZHEIMER
      S1   6255  (TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
?

S S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSGENIC) OR BIGENIC)
           6255  S1
          24565  COEXPRESSION
           4457  COEXPRESSING
         947980  DOUBLE
         195085  TRANSGENIC
           2203  DOUBLE(W)TRANSGENIC
            330  BIGENIC
      S2    286  S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W)
                 TRANSGENIC) OR BIGENIC)
?

S (SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR DRUG OR AGENT OR AGENTS
Processing
         121512  SCREEN
         682430  SCREENING
         176772  ASSAYED
          16176  ASSAYING
        1124916  DRUGS
        9758129  DRUG
        1660311  AGENT
        1762929  AGENTS
         524198  PHENOTYPE
             27  PHENOTYES
      S3 182903  (SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS
                 OR DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
?

S S2 AND S3
           286  S2
        182903  S3
      S4     0  S2 AND S3
?


Set     Items   Description
S1       6255   (TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
S2        286   S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSG-
              ENIC) OR BIGENIC)
S3     182903   (SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR
             DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
S4          0   S2 AND S3
?

S S2 AND (AGENT? OR DRUG?)
```

```
Processing
             286  S2
         3175869  AGENT?
        10102922  DRUG?
      S5      69  S2 AND (AGENT? OR DRUG?)
?

S S5 NOT PY>2000
              69  S5
        10825445  PY>2000
      S6       7  S5 NOT PY>2000
?

RD
      S7       5  RD  (unique items)
?

T S7/3,K/ALL

  7/3,K/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2007 Dialog. All rts. reserv.

12969837   PMID: 11113343
 Quantitative histological analysis of amyloid deposition in Alzheimer's
 double transgenic mouse brain.
  Wengenack T M; Whelan S; Curran G L; Duff K E; Poduslo J F
  Molecular Neurobiology Laboratory, Departments of Neurology and
Biochemistry/Molecular Biology, Mayo Clinic and Foundation, Rochester, MN
55905, USA.
  Neuroscience (UNITED STATES)    2000, 101 (4) p939-44, ISSN 0306-4522
--Print   Journal Code: 7605074
  Publishing Model Print
  Document type: Journal Article; Research Support, Non-U.S. Gov't
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed

 Quantitative histological analysis of amyloid deposition in Alzheimer 's
  double transgenic mouse brain.
  The development of transgenic mice has created new opportunities for
the generation of animal models of human neurodegenerative diseases where
previously there was no animal counterpart. The first successful
 transgenic mouse model of Alzheimer 's disease expressed increased
levels of mutant human amyloid precursor protein, exhibiting neuritic-type
amyloid...

... behavioral deficits at six to nine months of age. More recently, it was
shown that transgenic mice expressing both mutant human amyloid precursor
protein and presenilin 1 exhibit neuritic-type amyloid deposits and
behavioral deficits in as little as 12 weeks. This accelerated Alzheimer
 phenotype greatly reduces the time necessary to conduct preclinical drug
trials, as well as animal housing costs. The purpose of this study was to
quantify...

... of amyloid in five regions of the cortex and two regions of the
hippocampus of transgenic mice expressing amyloid precursor protein
(K670N, M671L) and presenilin 1 (M146L) mutations at various ages...

...the hippocampus. This was a function of increases in both deposit number
```

and size.This transgenic mouse provides an ideal animal model for evaluating the efficacy of potential therapeutic agents aimed at reducing amyloid deposition, such as inhibitors of amyloid fibril formation or secretase inhibitors.

7/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12709365 PMID: 10799751
Dominant negative effects of apolipoprotein E4 revealed in transgenic models of neurodegenerative disease.
Buttini M; Akeefe H; Lin C; Mahley R W; Pitas R E; Wyss-Coray T; Mucke L
Gladstone Institute of Neurological Disease University of California, P.O. Box 41900, San Francisco, CA 94141-9100, USA.
Neuroscience (UNITED STATES) 2000, 97 (2) p207-10, ISSN 0306-4522 --Print Journal Code: 7605074
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... cardiovascular and neurodegenerative disorders.(8,14) Apolipoprotein E4 is associated with an increased risk for Alzheimer 's disease(3,5) and poor clinical outcome after head injury or stroke.(11,16...

... remains unknown. To characterize the effects of human apolipoprotein E isoforms in vivo, we analysed transgenic Apoe knockout mice that express apolipoprotein E3 or E4 or both in the brain. Hemizygous...
... age-related and excitotoxin-induced neurodegeneration, whereas apolipoprotein E4 mice were not. Apolipoprotein E3/E4 bigenic mice were as susceptible to neurodegeneration as apolipoprotein E4 singly- transgenic mice. At eight months of age neurodegeneration was more severe in homozygous than in hemizygous...
...; Mice; Mice, Knockout; Mice, Transgenic; Microtubule-Associated Proteins--analysis--AN; Neurodegenerative Diseases--pathology--PA; Neuroprotective Agents ; Presynaptic Terminals--pathology--PA; Synaptophysin--analysis--AN
Chemical Name: Apolipoprotein E3; Apolipoprotein E4; Apolipoproteins E; Microtubule-Associated Proteins; Neuroprotective Agents ; Synaptophysin

7/3,K/3 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15904935 BIOSIS NO.: 200100076774
Can estrogen prevent cognitive decline and plaque formation in an experimental model of Alzheimer's disease?
AUTHOR: Miettinen R (Reprint); Puolivali J; Kalesnykas G; Heikkinen T; Iivonen S; Tapiola T; Tanila H
AUTHOR ADDRESS: Univ. Kuopio, Kuopio, Finland**Finland
JOURNAL: Society for Neuroscience Abstracts 26 (1-2): pAbstract No.-181.8 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000; 20001104
SPONSOR: Society for Neuroscience

ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Alzheimer 's disease (AD), the most common cause of age-associated dementia, is characterized especially by...

...estrogen replacement therapy can prevent plaque formation, and help to maintain cognitive functions in ovariectomized transgenic mice coexpressing familial AD-linked human presenilin 1 (A246E) and amyloid precursor protein (APPswe). Water maze test showed that i) transgenic mice were, in general, worse than wild type mice, ii) ovariectomy further impaired performance of the young transgenic mice, iii) which could be alleviated by estrogen treatment therapy. Biochemical analysis revealed that transgenic mice had exponentially increasing levels of both Abeta 1-40 and Abeta 1-42 peptides...

...estrogen treated mice. Bielschowsky silver staining showed that while the brains of 6 months old transgenic mice were usually devoid of plaques, those of 9 months old transgenic mice had a significant loads of plaques with different stages of maturation. In mice receiving estrogen replacement the plaque burden was attenuated compared with the non-treated transgenic mice. These findings provide evidence that estrogen replacement can diminish cognitive decline and beta-amyloid...
DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ...hormone- drug , nootropic- drug ;

7/3,K/4 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

10918398 EMBASE No: 2000413198
Astrocytic alterations in interleukin-6/soluble interleukin-6 receptor alpha double-transgenic mice
Brunello A.G.; Weissenberger J.; Kappeler A.; Vallan C.; Peters M.; Rose-John S.; Weis J.
Dr. J. Weis, Abtlg. Neuropathologie, Pathologisches Inst. der Universitat, Murtenstr. 31, CH-3012 Bern Switzerland
AUTHOR EMAIL: weisj@patho.unibe.ch
American Journal of Pathology (AM. J. PATHOL.) (United States) 2000, 157/5 (1485-1493)
CODEN: AJPAA ISSN: 0002-9440
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 66

Astrocytic alterations in interleukin-6/soluble interleukin-6 receptor alpha double - transgenic mice

...linked to several neurological disorders such as acquired immune deficiency syndrome dementia, multiple sclerosis, and Alzheimer 's disease. Central nervous system (CNS)-specific expression of IL-6 caused neurodegeneration, massive gliosis, and vascular proliferation in transgenic mice. However, the effects of systemically circulating IL-6 and its receptor IL-6Ralpha on...

...of either human IL-6 or human sIL-6Ralpha or both on the CNS of transgenic mice. Although IL-6 and sIL-6Ralpha single transgenic mice

were free of neurological disease, IL-6/sIL-6Ralpha doubletransgenic mice showed neurological signs...

...of IL-6/IL-6Ralpha such as liver damage and plasmacytomas. IL-6/sIL-6Ralpha transgenic mice exhibited massive reactive gliosis. Lack of signs of neuronal breakdown versus ample astrogliosis suggested...
DRUG DESCRIPTORS:
unclassified drug

7/3,K/5 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

07058223 EMBASE No: 1997340067
Accelerated amyloid deposition in the brains of transgenic mice coexpressing mutant presenilin 1 and amyloid precursor proteins
Borchelt D.R.; Ratovitski T.; Van Lare J.; Lee M.K.; Gonzales V.; Jenkins N.A.; Copeland N.G.; Price D.L.; Sisodia S.S.
D.R. Borchelt, Department of Pathology, Johns Hopkins School of Medicine, Baltimore, MD 21205 United States
Neuron (NEURON) (United States) 1997, 19/4 (939-945)
CODEN: NERNE ISSN: 0896-6273
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 51

Accelerated amyloid deposition in the brains of transgenic mice coexpressing mutant presenilin 1 and amyloid precursor proteins

...1 (PS1) and presenilin 2 (PS2), cause dementia in a subset of early-onset familial Alzheimer 's disease (FAD) pedigrees. In a variety of experimental in vitro and in vivo settings...

...the highly fibril-logenic Abeta1-42 peptides that are preferentially deposited in the brains of Alzheimer Disease (AD) patients. In this report, we demonstrate that transgenic animals that coexpress an FAD-linked human PSI variant (A246E) and a chimeric mouse/human...
DRUG DESCRIPTORS:
unclassified drug
?

Set	Items	Description
S1	6255	(TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
S2	286	S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSGENIC) OR BIGENIC)
S3	182903	(SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
S4	0	S2 AND S3
S5	69	S2 AND (AGENT? OR DRUG?)
S6	7	S5 NOT PY>2000
S7	5	RD (unique items)

?

S (TRANSGENIC OR DROSOPHILA OR ELEGANS) AND (ALZHEIMER)
195085 TRANSGENIC
185235 DROSOPHILA
52105 ELEGANS
180222 ALZHEIMER

```
      S8    8377  (TRANSGENIC OR DROSOPHILA OR ELEGANS) AND (ALZHEIMER)
?

S S8 AND (COEXPRESSING OR COEXPRESSING OR BIGENIC OR (DOUBLE (W) TRANSGENIC))
            8377  S8
            4457  COEXPRESSING
            4457  COEXPRESSING
             330  BIGENIC
          947980  DOUBLE
          195085  TRANSGENIC
            2203  DOUBLE(W)TRANSGENIC
      S9     313  S8 AND (COEXPRESSING OR COEXPRESSING OR BIGENIC OR
                  (DOUBLE (W) TRANSGENIC))
?


Set     Items   Description
S1       6255   (TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
S2        286   S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSG-
              ENIC) OR BIGENIC)
S3     182903   (SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR
              DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
S4          0   S2 AND S3
S5         69   S2 AND (AGENT? OR DRUG?)
S6          7   S5 NOT PY>2000
S7          5   RD  (unique items)
S8       8377   (TRANSGENIC OR DROSOPHILA OR ELEGANS) AND (ALZHEIMER)
S9        313   S8 AND (COEXPRESSING OR COEXPRESSING OR BIGENIC OR (DOUBLE
              (W) TRANSGENIC))
?

S S9 AND S3
             313  S9
          182903  S3
     S10       1  S9 AND S3
?

T S10/3,K/ALL

  10/3,K/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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18024082   BIOSIS NO.: 200400394871
 Cell cultures from animal models of Alzheimer's disease as a tool for
 faster screening and testing of drug efficacy
AUTHOR: Trinchese Fabrizio; Liu Shumin; Ninan Ipe; Puzzo Daniela; Jacob
  Joel P; Arancio Ottavio (Reprint)
AUTHOR ADDRESS: Dept PsychiatSch Med, NYU, 550 1St Ave, New York, NY,
  10016, USA**USA
AUTHOR E-MAIL ADDRESS: oa1@columbia.edu
JOURNAL: Journal of Molecular Neuroscience  24 (1): p15-21 2004 2004
MEDIUM: print
ISSN: 0895-8696 _(ISSN online)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

 Cell cultures from animal models of  Alzheimer 's disease as a tool for
 faster  screening  and testing of  drug  efficacy
```

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ABSTRACT: Approximately 2 million people in the United States suffer from
  Alzheimer 's disease (AD), which is the most common cause of chronic
  dementia among the aging population. During the last 7 yr, excellent
  opportunities to screen drugs against AD have been provided by animal
  models of the disease. Because even in the...

...second month, it has been necessary to wait at least until that age to
  inject drugs into the animal to assess whether they prevent, reduce, or
  revert synaptic impairment, plaque formation...

...reproducible cultured cell system from animal models of AD or
  Abeta-associated diseases, for the screening and testing of compounds
  for the treatment and therapy of AD or Abeta-associated diseases.
DESCRIPTORS:
  ...ORGANISMS: immature, animal model, double transgenic , strain-APP
    , strain-APP-PS1, strain-PS1
  DISEASES: Alzheimer 's disease...
  MESH TERMS: Alzheimer Disease (MeSH...
?


Set     Items   Description
S1       6255   (TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
S2        286   S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSG-
            ENIC) OR BIGENIC)
S3     182903   (SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR
            DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
S4          0   S2 AND S3
S5         69   S2 AND (AGENT? OR DRUG?)
S6          7   S5 NOT PY>2000
S7          5   RD (unique items)
S8       8377   (TRANSGENIC OR DROSOPHILA OR ELEGANS) AND (ALZHEIMER)
S9        313   S8 AND (COEXPRESSING OR COEXPRESSING OR BIGENIC OR (DOUBLE
            (W) TRANSGENIC))
S10         1   S9 AND S3
?

S S8 AND S3
            8377  S8
          182903  S3
     S11     111  S8 AND S3
?

S S11 AND (MODIFER OF (X (W) DEFICIENCIES))
             111  S11
               0  MODIFER OF (X
               0  DEFICIENCIES)
               0  MODIFER OF (X(W)DEFICIENCIES)
     S12       0  S11 AND (MODIFER OF (X (W) DEFICIENCIES))
?


Set     Items   Description
S1       6255   (TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
S2        286   S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSG-
            ENIC) OR BIGENIC)
S3     182903   (SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR
            DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
S4          0   S2 AND S3
```

```
S5          69   S2 AND (AGENT? OR DRUG?)
S6           7   S5 NOT PY>2000
S7           5   RD  (unique items)
S8        8377   (TRANSGENIC OR DROSOPHILA OR ELEGANS) AND (ALZHEIMER)
S9         313   S8 AND (COEXPRESSING OR COEXPRESSING OR BIGENIC OR (DOUBLE
                 (W) TRANSGENIC))
S10          1   S9 AND S3
S11        111   S8 AND S3
S12          0   S11 AND (MODIFER OF (X (W) DEFICIENCIES))
?

S S11 NOT PY>2000
              111  S11
         10825445  PY>2000
     S13      18  S11 NOT PY>2000
?

RD
     S14      13  RD  (unique items)
?


Set     Items   Description
S1       6255   (TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
S2        286   S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSG-
                ENIC) OR BIGENIC)
S3     182903   (SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR
                DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
S4          0   S2 AND S3
S5         69   S2 AND (AGENT? OR DRUG?)
S6          7   S5 NOT PY>2000
S7          5   RD  (unique items)
S8       8377   (TRANSGENIC OR DROSOPHILA OR ELEGANS) AND (ALZHEIMER)
S9        313   S8 AND (COEXPRESSING OR COEXPRESSING OR BIGENIC OR (DOUBLE
                (W) TRANSGENIC))
S10         1   S9 AND S3
S11       111   S8 AND S3
S12         0   S11 AND (MODIFER OF (X (W) DEFICIENCIES))
S13        18   S11 NOT PY>2000
S14        13   RD  (unique items)
?

S S14 NOT (S7 OR S10)
              13  S14
               5  S7
               1  S10
     S15      13  S14 NOT (S7 OR S10)
?

T S15/3,K/ALL

  15/3,K/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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12972769   PMID: 11114162
 spr-2, a suppressor of the egg-laying defect caused by loss of sel-12
 presenilin in Caenorhabditis elegans, is a member of the SET protein
 subfamily.
  Wen C; Levitan D; Li X; Greenwald I
```

Department of Biochemistry and Molecular Biophysics, Howard Hughes Medical Institute, Columbia University, New York, NY 10032, USA.
Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Dec 19 2000, 97 (26) p14524-9, ISSN 0027-8424 --Print Journal Code: 7505876
Contract/Grant No.: NS35556; NS; NINDS
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... suppressor of the egg-laying defect caused by loss of sel-12 presenilin in Caenorhabditis elegans , is a member of the SET protein subfamily.

Presenilin plays critical roles in the genesis of Alzheimer 's disease and in LIN-12/Notch signaling during development. Here, we describe a screen for genes that influence presenilin level or activity in Caenorhabditis elegans . We identified four spr (suppressor of presenilin) genes by reverting the egg-laying defective phenotype caused by a null allele of the sel-12 presenilin gene. We analyzed the spr...

... some detail. We show that loss of spr-2 activity suppresses the egg-laying defective phenotype of different sel-12 alleles and requires activity of the hop-1 presenilin gene, suggesting...

Descriptors: *Caenorhabditis elegans Proteins; *Helminth Proteins --genetics--GE; *Helminth Proteins--metabolism--ME; *Membrane Proteins --metabolism--ME; *Nuclear Proteins...
; Alleles; Amino Acid Sequence; Animals; Animals, Genetically Modified; Base Sequence; Caenorhabditis elegans ; Cell Nucleus--metabolism--ME; Chromosomal Proteins, Non-Histone; Cloning, Molecular; DNA, Helminth; Gene Expression Regulation...

Chemical Name: Caenorhabditis elegans Proteins; Chromosomal Proteins, Non-Histone; DNA, Helminth; Helminth Proteins; Hop-1 protein, C elegans ; Luminescent Proteins; Membrane Proteins; Nuclear Proteins; Proteins; SEL-12 protein, C elegans ; SET protein, human; Spr-2 protein, C elegans ; Transcription Factors; template activating factor-I; Green Fluorescent Proteins

15/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12201778 PMID: 10621945
Neuroprotective approaches in experimental models of beta-amyloid neurotoxicity: relevance to Alzheimer's disease.

Harkany T; Hortobagyi T; Sasvari M; Konya C; Penke B; Luiten P G; Nyakas C
Central Research Division of Clinical and Experimental Laboratory Medicine, Haynal Imre University of Health Sciences, Budapest, Hungary. harkanyt@biol.rug.nl
Progress in neuro-psychopharmacology & biological psychiatry (ENGLAND) Aug 1999, 23 (6) p963-1008, ISSN 0278-5846--Print Journal Code: 8211617
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't; Review
Languages: ENGLISH
Main Citation Owner: NLM

Record type: MEDLINE; Completed

Neuroprotective approaches in experimental models of beta-amyloid neurotoxicity: relevance to Alzheimer 's disease.

1. beta-Amyloid peptides (A beta s) accumulate abundantly in the Alzheimer 's disease (AD) brain in areas subserving information acquisition and processing, and memory formation. A...

... circulation were developed in order to investigate the effects of synthetic A beta s, whereas transgenic models provided insight into the distinct molecular steps of pathological APP cleavage. 3. The hippocampus, caudate putamen, amygdala and neocortex all formed primary targets of acute neurotoxicity screening , but functional consequences of A beta infusions were primarily demonstrated following either intracerebroventricular or basal...

... as vitamin E or vitamin C, attenuated A beta toxicity with high efficacy. Interestingly, combined drug treatments did not necessarily result in additive enhanced neuroprotection. 7. Similarly to the blockade of...

... manipulation of voltage-dependent Ca(2+)-channels, serotonergic IA or adenosine A1 receptors, and by drugs eliciting membrane hyperpolarization or indirect blockade of Ca(2+)-mediated intracellular consequences of intracerebral A...

Descriptors: *Alzheimer Disease--drug therapy--DT; *Amyloid beta-Protein--toxicity--TO; *Neuroprotective Agents--therapeutic use--TU; Alzheimer Disease--pathology--PA; Amyloid beta-Protein--antagonists and inhibitors--AI; Animals; Humans; Neuroprotective Agents--pharmacology...

15/3,K/3 (Item 3 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

11269071 PMID: 9062918

Rapid drug screening for Alzheimer's.

Dobeli H

Nature biotechnology (UNITED STATES) Mar 1997, 15 (3) p223-4, ISSN 1087-0156--Print Journal Code: 9604648

Publishing Model Print

Document type: News

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Rapid drug screening for Alzheimer 's.

Descriptors: *Alzheimer Disease--drug therapy--DT; Alzheimer Disease --genetics--GE; Amyloid beta-Protein--antagonists and inhibitors--AI; Animals; Chromosome Mapping; Disease Models, Animal; Drug Design; Humans; Mice; Mice, Transgenic

15/3,K/4 (Item 4 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

09127730 PMID: 1367956

Mouse models of human diseases.

Westphal H

Laboratory of Mammalian Genes and Development, National Institute of Child Health and Human Development, National Institute of Health, Bethesda, MD 20892.
Current opinion in biotechnology (ENGLAND) Dec 1991, 2 (6) p830-3, ISSN 0958-1669--Print Journal Code: 9100492
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Cancer, poliomyelitis, Alzheimer 's and Gaucher disease, a seemingly disparate array of disorders, have become the target of powerful genetic analysis and drug screening protocols, using mouse strains that have been genetically altered to serve as models for understanding...
Descriptors: *Alzheimer Disease--genetics--GE; *Disease Models, Animal; *Neoplasms, Experimental--genetics--GE; *Poliomyelitis--genetics--GE; Animals; Humans; Mice; Mice, Transgenic

15/3,K/5 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14660351 BIOSIS NO.: 199800454598
Alzheimer's disease and risk factors
AUTHOR: Wen G Y (Reprint)
AUTHOR ADDRESS: New York State Inst. Basic Res. Developmental Disabilities, 10450 Forest Hill Road, Staten Island, NY 10314, USA**USA
JOURNAL: Journal of Food and Drug Analysis 6 (2): p465-476 June, 1998 1998
MEDIUM: print
ISSN: 1021-9498
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

Alzheimer 's disease and risk factors

ABSTRACT: Alzheimer 's disease (AD) strikes more than 3 million people in the United States and 17...

...in those individuals with AD alone. This observation has provided the rationale for generating the transgenic , mouse models of AD. The treatment of AD with drugs such as Tacrine or Aricept represents a certain degree of success (not a cure), but the transgenic AD mice may facilitate the development and screening of more effective new drugs for AD.
DESCRIPTORS:
...ORGANISMS: animal model, transgenic
...DISEASES: Alzheimer 's disease
...MESH TERMS: Alzheimer Disease (MeSH

15/3,K/6 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

10943313 EMBASE No: 2000431975
Transgenic mouse models and human neurodegenerative disorders
Deng H.-X.; Siddique T.

Dr. T. Siddique, Department of neurology, Northwestern University Medical Sch., Tarry 13-715, 303 E Chicago Ave, Chicago, IL 60611 United States
AUTHOR EMAIL: t-siddique@nwu.edu
Archives of Neurology (ARCH. NEUROL.) (United States) 2000, 57/12 (1695-1702)
CODEN: ARNEA ISSN: 0003-9942
DOCUMENT TYPE: Journal; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 36

Transgenic mouse models and human neurodegenerative disorders

The development of new methods for manipulating the mouse genome by transgenic and gene-targeting technologies has dramatically increased our ability to create mouse models for human...

...understanding of the pathogenesis of some human diseases and are beginning to be used in screening of therapeutic agents . In this review, we outline 2 basic techniques that are most frequently used to alter...
MEDICAL DESCRIPTORS:
transgenic mouse; gene targeting; technique; genome; Alzheimer disease; prion disease; nonhuman; mouse; animal experiment; animal model; conference paper; priority journal

15/3,K/7 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

10821561 EMBASE No: 2000303654
Alzheimer's disease: Transgenic mouse models and drug assessment
Yu P.; Oberto G.
P. Yu, General Toxicology I Unit, Istituto di Ricerche Biomediche, Via Ribes I, 10010 Colleretto Giacosa (TO) Italy
Pharmacological Research (PHARMACOL. RES.) (United Kingdom) 2000, 42/2 (107-114)
CODEN: PHMRE ISSN: 1043-6618
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 92

Alzheimer 's disease: Transgenic mouse models and drug assessment

Alzheimer 's disease (AD), characterized by neuritic plaques and neurofibrillary tangles of the brain, is experienced...

...are closely linked with AD and are located on chromosomes 21, 19, 14 and 1. Transgenic technology enables the development of animal models for research into this human disease. Recently reported transgenic AD mouse models, which express AD-related mutant human genes, develop some significant aspects of...
MEDICAL DESCRIPTORS:
* Alzheimer disease--etiology--et; *transgene
transgenic mouse; senile plaque; neurofibrillary tangle; aging; senile dementia; genetic linkage; gene mutation; drug screening ; nonhuman; mouse; animal experiment; animal model; review; priority journal

15/3,K/8 (Item 3 from file: 73)

DIALOG(R)File 73:EMBASE

07549392 EMBASE No: 1999041424
Transgenic animals and cell lines for screening drugs effective for the treatment or prevention of Alzheimer's disease
Expert Opinion on Therapeutic Patents (EXPERT OPIN. THER. PAT.) (United Kingdom) 1999, 9/2 (201-204)
CODEN: EOTPE ISSN: 1354-3776
DOCUMENT TYPE: Journal; Short Survey
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 16

Transgenic animals and cell lines for screening drugs effective for the treatment or prevention of Alzheimer 's disease

Overexpression of neuronal thread protein has been reported in the Alzheimer 's disease (AD) brain, reflecting the widespread cortical neuritic sprouting characteristic of AD; this overexpression...

...AD produced by neuronal thread protein overexpression, and that these models may be useful for screening potential drug candidates for the treatment of AD.
MEDICAL DESCRIPTORS:
* transgenic animal; *cell line; * Alzheimer disease
drug screening ; nerve sprouting; protein expression; patent; genetic transfection; nonhuman; short survey

15/3,K/9 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

07474810 EMBASE No: 1998407304
Recent advances in transgenic model development for Alzheimer's disease
Sommer B.
B. Sommer, Nervous System Research, Novartis Pharma AG, CH-4002 Basel Switzerland
Expert Opinion on Investigational Drugs (EXPERT OPIN. INVEST. DRUGS) (United Kingdom) 1998, 7/12 (2017-2025)
CODEN: EOIDE ISSN: 1354-3784
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 60

Recent advances in transgenic model development for Alzheimer 's disease

The lack of a small animal model that represents major features of Alzheimer 's disease has long been considered a major handicap for research and drug development. Transgenic technology has been used to introduce potential pathological start points as well as established genetic...

...trigger pathogenesis in a small animal model. This review describes various approaches, discusses the available transgenic mouse models and compares their similarities and differences, and their applicability for the testing of...
MEDICAL DESCRIPTORS:
* alzheimer disease--etiology--et
animal model; pathogenesis; transgenic mouse; drug screening ;

nonhuman; mouse; review

15/3,K/10 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

07162211 EMBASE No: 1998050358
Experimental and clinical methods in the development of anti-Alzheimer drugs
Allain H.; Bentue-Ferrer D.; Zekri O.; Schuck S.; Lebreton S.; Reymann J.M.
O. Zekri, Unite de Pharmacoepidemiologie, Faculte de Medecine, Avenue du Pr. Leon Bernard, 35043 Rennes France
Fundamental and Clinical Pharmacology (FUNDAM. CLIN. PHARMACOL.) (France) 1998, 12/1 (13-29)
CODEN: FCPHE ISSN: 0767-3981
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 139

Experimental and clinical methods in the development of anti- Alzheimer drugs

Methodology used for the development of anti- Alzheimer 's disease (AD) drugs raises specific problems which are rarely examined in the literature. While...
...drugs. During preclinical studies, aged or lesioned animals are mainly useful for symptomatic drugs, whereas transgenic models or neurodegeneration-induced techniques would probably lead to etiopathogenic drugs potentially slowing down the...
MEDICAL DESCRIPTORS:
* alzheimer disease--drug therapy--dt; * alzheimer disease--etiology--et; *cholinergic system
drug development; methodology; animal model; transgenic animal; psychometry; electrophysiology; cognition; image analysis; practice guideline; drug screening ; senile plaque--drug therapy--dt; senile plaque--etiology--et; senile plaque--prevention--pc; mutation; heredity...

15/3,K/11 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

06913073 EMBASE No: 1997197515
Alzheimer's disease and related Dementias: Prospects for treatment
Williams M.; Davis R.E.
M. Williams, NUDRD 464, Abbott Laboratories, 100 Abbott Park Road, Abbott Park, IL, 60064-3500 United States
AUTHOR EMAIL: mike.williams@abbott.com
Expert Opinion on Investigational Drugs (EXPERT OPIN. INVEST. DRUGS) (United Kingdom) 1997, 6/6 (735-757)
CODEN: EOIDE ISSN: 1354-3784
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 62

Alzheimer 's disease and related Dementias: Prospects for treatment

Alzheimer 's disease (AD) represents a major challenge to healthcare

costs and to academic and pharmaceutical...

...environmental, may contribute to the pathophysiology of AD unrelated to familial cohorts. A newly developed transgenic mouse model and a broader appreciation of the multifactorial nature of this complex, chronic disease
...
MEDICAL DESCRIPTORS:
* alzheimer disease--etiology--et; *dementia--etiology--et
drug efficacy; drug screening ; estrogen therapy; pathogenesis; review; risk assessment; treatment outcome

15/3,K/12 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

06040428 EMBASE No: 1995070695
Alzheimer's disease: Fundamental and therapeutic aspects
Schorderet M.
Departement de Pharmacologie, Centre Medical Universitaire, 1 rue Michel Servet,CH-1211 Geneve 4 Switzerland
Experientia (EXPERIENTIA) (Switzerland) 1995, 51/2 (99-105)
CODEN: EXPEA ISSN: 0014-4754
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Alzheimer 's disease: Fundamental and therapeutic aspects

Alzheimer 's disease is the most common type of progressive and debilitating dementia affecting aged people...

...and memory deficits. Several compounds are being tested in attempts to prevent and/or cure Alzheimer 's disease, including tacrine, which has very modest efficacy in a sub-group of patients...

...for neurodegenerative diseases induced by multiple endogenous and/or exogenous factors. The recent use of transgenic mice, in parallel with other genetic, biochemical and neurobiological systems, in vivo and/or in
...

...cell cultures), should accelerate the discovery and development of specific drugs for the treatment of Alzheimer 's disease.
MEDICAL DESCRIPTORS:
* alzheimer disease--drug therapy--dt; * alzheimer disease--epidemiology --ep; * alzheimer disease--etiology--et; * alzheimer disease--prevention --pc
...pc; cognitive defect--drug therapy--dt; cognitive defect--etiology--et; dementia--etiology--et; drug efficacy; drug screening ; gastrointestinal symptom--side effect--si; genetic linkage; ginkgo biloba; hippocampus; human; liver toxicity--side effect...

...neurologic disease--side effect--si; neurotransmission; nonhuman; oral drug administration; protein phosphorylation; review; senile plaque; transgenic mouse

15/3,K/13 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

05672882 EMBASE No: 1994080572

Reverse genetics of the mouse central nervous system: Targeted genetic analysis of neuropeptide function and reverse genetic screens for genes involved in human neurodegenerative disease

Davies R.W.; Gallagher E.J.; Savioz A.
Robertson Institute of Biotechnology, Department of Genetics, University of Glasgow, 54 Dunbarton Road,Glasgow G11 6NU United Kingdom

Progress in Neurobiology (PROG. NEUROBIOL.) (United Kingdom) 1994, 42/2 (319-331)
CODEN: PGNBA ISSN: 0301-0082
DOCUMENT TYPE: Journal; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

...make chimaeric mice, some of which transmit the in vitro mutation via the germline to transgenic offspring. The phenotype of complete loss-of-function mutations (gene knock-outs) can be studied at molecular, cell...

...technological improvements makes targeted mutation of a number of genes possible. This allows reverse genetic screening to be undertaken for genes involved in particular neurobiological phenomena: genes are identified on the...

...criteria (e.g. expression pattern), and gene-targeting used to check their relevance to a phenotype . Neurodegenerative disease is an important aspect of the human phenotype . In both Parkinson's disease and Alzheimer 's disease particular neuronal cell-types or particular brain regions are much more susceptible than...

...area of mouse ventral midbrain. Candidate genes with localised expression patterns are identified by differential screening and differential display analysis followed by in situ hybridisation. The effects of targeted mutations in...

MEDICAL DESCRIPTORS:
alzheimer disease--congenital disorder--cn; alzheimer disease--etiology --et; animal tissue; conference paper; human; mouse; mutant; nonhuman; parkinson disease--etiology--et...
?

Set	Items	Description
S1	6255	(TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
S2	286	S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSG-ENIC) OR BIGENIC)
S3	182903	(SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
S4	0	S2 AND S3
S5	69	S2 AND (AGENT? OR DRUG?)
S6	7	S5 NOT PY>2000
S7	5	RD (unique items)
S8	8377	(TRANSGENIC OR DROSOPHILA OR ELEGANS) AND (ALZHEIMER)
S9	313	S8 AND (COEXPRESSING OR COEXPRESSING OR BIGENIC OR (DOUBLE (W) TRANSGENIC))
S10	1	S9 AND S3
S11	111	S8 AND S3
S12	0	S11 AND (MODIFER OF (X (W) DEFICIENCIES))
S13	18	S11 NOT PY>2000
S14	13	RD (unique items)
S15	13	S14 NOT (S7 OR S10)

?

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S S14 AND (HAR38 OR DCREBA OR DCREBB OR ADAPTIN OR GARNET OR SHI OR MAM OR BIB)
              13  S14
               3  HAR38
               1  DCREBA
               0  DCREBB
            1133  ADAPTIN
            4115  GARNET
            2552  SHI
            2859  MAM
             476  BIB
     S16       0  S14 AND (HAR38 OR DCREBA OR DCREBB OR ADAPTIN OR GARNET
                  OR SHI OR MAM OR BIB)
?

S (HAR38 OR DCREBA OR DCREBB OR ADAPTIN OR GARNET OR BIB) (S) ALZHEIMER)
>>>Unmatched parentheses
?

S (HAR38 OR DCREBA OR DCREBB OR ADAPTIN OR GARNET OR BIB OR GARNET OR SHI OR MAM) (S
               3  HAR38
               1  DCREBA
               0  DCREBB
            1133  ADAPTIN
            4115  GARNET
             476  BIB
            4115  GARNET
            2552  SHI
            2859  MAM
          180222  ALZHEIMER
     S17      20  (HAR38 OR DCREBA OR DCREBB OR ADAPTIN OR GARNET OR BIB OR
                  GARNET OR SHI OR MAM) (S) (ALZHEIMER)
?

S S17 AND (VECTOR OR GENE)
              20  S17
          340293  VECTOR
         3018808  GENE
     S18       5  S17 AND (VECTOR OR GENE)
?

RD
     S19       3  RD  (unique items)
?

T S19/3,K/ALL

  19/3,K/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2007 Dialog. All rts. reserv.

12581467   PMID: 10600649
 Intrinsic alcohol dehydrogenase and hydroxysteroid dehydrogenase
 activities of human mitochondrial short-chain L-3-hydroxyacyl-CoA
 dehydrogenase.
  He X Y; Yang Y Z; Schulz H; Yang S Y
  Department of Pharmacology, New York State Institute for Basic Research
in Developmental Disabilities, Staten Island, NY 10314, USA.
  Biochemical journal (ENGLAND) Jan 1 2000, 345 Pt 1 p139-43, ISSN
0264-6021--Print  Journal Code: 2984726R
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Contract/Grant No.: AG04220; AG; NIA; DK47392; DK; NIDDK; HL30847; HL; NHLBI
Publishing Model Print
Document type: In Vitro; Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... less than those reported for endoplasmic-reticulum-associated amyloid beta-peptide-binding protein (ERAB) [Yan, Shi , Zhu, Fu, Zhu, Zhu, Gibson, Stern, Collison, Al-Mohanna et al. (1999) J. Biol. Chem...
...catalytic properties should be identical. The recombinant SCHAD has been confirmed to be the right gene product and not a mutant variant. Steady-state kinetic measurements and quantitative analyses reveal that...

...important multifunctional enzyme paves the way for exploring its role(s) in the pathogenesis of Alzheimer 's disease.

19/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12517702 PMID: 10461542
Damage and repair of nerve cell DNA in toxic stress.
Kisby G E; Kabel H; Hugon J; Spencer P
Center for Research on Occupational and Environmental Toxicology, School of Medicine, Oregon Health Sciences University, Portland 97201, USA. kisby@ohsu.edu
Drug metabolism reviews (UNITED STATES) Aug 1999, 31 (3) p589-618, ISSN 0360-2532--Print Journal Code: 0322067
Contract/Grant No.: NS19611; NS; NINDS
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... A strong candidate is the cycad plant genotoxin cycasin, the beta-D-glucoside of methylazoxymethanol (MAM). We propose that prenatal or postnatal exposure to low levels of cycasin/ MAM may damage neuronal DNA, compromise DNA repair, perturb neuronal gene expression, and irreversibly alter cell function to precipitate a slowly evolving disease ("slow-toxin" hypothesis...

... 1. DNA from postmitotic rodent central nervous system neurons is particularly sensitive to damage by MAM . 2. MAM reduces DNA repair in human and rodent neurons, whereas DNA-repair inhibitors potentiate MAM -induced DNA damage and toxicity in mature rodent nervous tissue. 3. Human neurons (SY5Y neuroblastoma) that are deficient in DNA repair are susceptible to MAM -induced cytotoxicity and DNA damage, whereas overexpression of DNA repair in similar cells is protective. 4. MAM alters gene expression in SY5Y human neuroblastoma cells and, in the presence of DNA damage and reduced...

...mRNA in rat primary neurons; the corresponding protein (TAU) is elevated in ALS/PDC and Alzheimer 's disease. These findings support a direct relationship between MAM -induced DNA damage and neurotoxicity and suggest

the genotoxin may operate in a similar manner...

...repair is reduced in the brain of subjects with western Pacific ALS/PDC, ALS, and Alzheimer 's disease, which would increase the susceptibility of brain tissue to DNA damage by endogenous...

... underway using DNA-repair proficient and deficient neuronal cell cultures and mutant mice to explore gene -environment interplay with respect to MAM treatment, DNA damage, and DNA repair, and the age-related appearance of neurobehavioral and neuropathological...
; Animals; Carcinogens--toxicity--TO; DNA Repair--physiology--PH; Gene Expression--drug effects--DE; Gymnosperms--toxicity--TO; Humans; Methylazoxymethanol Acetate--analogs and derivatives--AA; Methylazoxymetha nol...

19/3,K/3 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17356563 BIOSIS NO.: 200300315282
GENE EXPRESSION PROFILING OF THE DEVELOPING BRAIN FOLLOWING TREATMENT WITH METHYLAZOXYMETHANOL (MAM).
AUTHOR: Kisby G E (Reprint); Sproles D (Reprint); Pattee P; Nagalla S R
AUTHOR ADDRESS: Ctr Res Occup and Enviro Toxicol, Oregon Hlth and Sci Univ, Portland, OR, USA**USA
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner 2002 pAbstract No. 597.4 2002 2002
MEDIUM: cd-rom
CONFERENCE/MEETING: 32nd Annual Meeting of the Society for Neuroscience Orlando, Florida, USA November 02-07, 2002; 20021102
SPONSOR: Society for Neuroscience
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

GENE EXPRESSION PROFILING OF THE DEVELOPING BRAIN FOLLOWING TREATMENT WITH METHYLAZOXYMETHANOL (MAM).

ABSTRACT: The genotoxin methylazoxymethanol (MAM) is a developmental neurotoxin and etiological factor for a progressive neurological disorder in the western Pacific with features of ALS, Parkinson's disease, and an Alzheimer -like dementia (ALS/PDC). The mechanism of MAM induced acute or chronic brain injury is poorly understood. To determine the role of gene expression changes in MAM induced brain injury, 3-day old C57BL6 mice were administered saline or a sub-lethal dose of MAM (43 mg/kg, s.c.), and 1, 8, 15, and 22 days later RNA isolated...

...of apprx26,000 mouse sequence verified clones. Preliminary data analysis showed region-specific changes in gene expression. Cerebellum, the most affected region, had a high number of differentially expressed genes with ...

...12-fold) and 24 down-regulated (3 to 9-fold) genes after 1 day of MAM treatment. Significant changes were also detected in the cerebral cortex of the same mice, a brain region reportedly unaffected by the genotoxin. More importantly, minimal gene overlap was observed between the cerebral cortex and cerebellum of mice treated for 1 day with MAM . These studies demonstrate that gene expression in both affected and unaffected brain regions is regulated in a distinct manner by the

```
  genotoxin  MAM  and this may explain its ability to induce acute and
  chronic brain tissue injury.
DESCRIPTORS:
  CHEMICALS & BIOCHEMICALS:    ... gene --
  METHODS & EQUIPMENT:  gene  expression profiling...
?


Set      Items   Description
S1        6255   (TRANSGENIC OR DROSOPHILA OR ELEGANS) (S) (ALZHEIMER)
S2         286   S1 AND (COEXPRESSION OR COEXPRESSING OR (DOUBLE (W) TRANSG-
              ENIC) OR BIGENIC)
S3      182903   (SCREEN OR SCREENING OR ASSAYED OR ASSAYING) (S) (DRUGS OR
              DRUG OR AGENT OR AGENTS OR PHENOTYPE OR PHENOTYES)
S4           0   S2 AND S3
S5          69   S2 AND (AGENT? OR DRUG?)
S6           7   S5 NOT PY>2000
S7           5   RD  (unique items)
S8        8377   (TRANSGENIC OR DROSOPHILA OR ELEGANS) AND (ALZHEIMER)
S9         313   S8 AND (COEXPRESSING OR COEXPRESSING OR BIGENIC OR (DOUBLE
              (W) TRANSGENIC))
S10          1   S9 AND S3
S11        111   S8 AND S3
S12          0   S11 AND (MODIFER OF (X (W) DEFICIENCIES))
S13         18   S11 NOT PY>2000
S14         13   RD  (unique items)
S15         13   S14 NOT (S7 OR S10)
S16          0   S14 AND (HAR38 OR DCREBA OR DCREBB OR ADAPTIN OR GARNET OR
              SHI OR MAM OR BIB)
S17         20   (HAR38 OR DCREBA OR DCREBB OR ADAPTIN OR GARNET OR BIB OR -
              GARNET OR SHI OR MAM) (S) (ALZHEIMER)
S18          5   S17 AND (VECTOR OR GENE)
S19          3   RD  (unique items)
?

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            $8.68    2.553 DialUnits File155
               $1.76  8 Type(s) in Format  3
            $1.76  8 Types
    $10.44  Estimated cost File155
           $10.75    1.791 DialUnits File5
               $9.20  4 Type(s) in Format  3
            $9.20  4 Types
    $19.95  Estimated cost File5
           $50.37    4.232 DialUnits File73
              $33.00  10 Type(s) in Format  3
           $33.00  10 Types
    $83.37  Estimated cost File73
            OneSearch, 3 files,  8.577 DialUnits FileOS
     $7.20  INTERNET
   $120.96  Estimated cost this search
   $121.98  Estimated total session cost   8.853 DialUnits
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Refine Search

Search Results -

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GENE	364989
GENES	158146
VECTOR	432281
VECTORS	228552
(8 SAME (VECTOR OR GENE)).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	36
(L8 SAME (GENE OR VECTOR)).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	36

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IBM Technical Disclosure Bulletins

Search: L9

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Thursday, June 21, 2007 **Purge Queries** Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=AND			
L9	L8 same (gene or vector)	36	L9
L8	(har38 or dCrebA or dCrebB or adaptin or garnet or shi or mam or bib) same (Alzheimer)	293	L8
L7	L5 not L6	25	L7
L6	L5 and (APPL or APP or PSN or presenilin or PS1)	181	L6

L5	L4 and L3	206	L5
L4	(screen or screening or assayed or assaying) same (drug or phenotype or agent)	139458	L4
L3	L2 and (coexpression or (double adj transgenic) or coexpressing)	258	L3
L2	(Transgenic or Drosophila or elegans) same (Alzheimer)	2469	L2
L1	Greenspan-Ralph-J$.in.	9	L1

END OF SEARCH HISTORY

PALM INTRANET

Day : Thursday
Date: 6/21/2007

Time: 13:48:00

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	**First Name**	
Greenspan	Ralph	Search

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Date: 6/21/2007

Time: 13:48:00

Inventor Name Search

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Last Name	**First Name**	
Edelman	Gerald	Search

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